UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/727,195	12/03/2003	Carmen V. Pepicelli	HUIP-P02-032	6922

28120 7590 08/03/2006

FISH & NEAVE IP GROUP
ROPES & GRAY LLP
ONE INTERNATIONAL PLACE
BOSTON, MA 02110-2624

EXAMINER	
HOWARD, ZACHARY C	

ART UNIT	PAPER NUMBER
1646	

DATE MAILED: 08/03/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/727,195	PEPICELLI ET AL.	
	Examiner	Art Unit	
	Zachary C. Howard	1646	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 May 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-17 and 19-26* is/are pending in the application.

4a) Of the above claim(s) *5-17* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-4,19-26* is/are rejected.

7)☒ Claim(s) *1,2 and 19-22* is/are objected to.

8)☒ Claim(s) *1-17 and 19-26* are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *16 May 2003* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Status of Application, Amendments and/or Claims

An Office Action with a Non-Final Rejection of the claims was mailed 9/20/05. Applicants' 1/23/06 response included amendments to the claims (pg 4-8) and remarks (pg 9-15). These claim amendments were entered in the subsequent Office Action, mailed 4/17/06 and necessitated a further Restriction Requirement (species election only) due to the addition of several species to the claims. Applicants' response to the Restriction Requirement was received 5/19/06, along with a copy of the claims presented for the Examiner's convenience (no claim amendments were made 5/19/06).

Claims 1-17 and 19-26 are pending.

This application contains claims 5-17 drawn to an invention nonelected with traverse in Applicant's response filed 6/27/05. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Election/Restrictions

Applicant's election with traverse of the species of "small organic molecules" in the reply filed on 5/19/06 is acknowledged.

The traversal is on the ground(s) that the identified species correspond to overlapping subject matter, and submit that searches of the identified species are co-extensive. Applicants argue that claims directed to the use of various species of ptc therapeutics can be examined simultaneously without significant additional burden.

This is found persuasive *in part*. In the 4/17/06 Restriction Requirement (species election), Applicants were required to elect a species of ptc therapeutic selected from: small organic molecule, protein kinase A inhibitor or ptc antisense construct. The specification does not provide a definition of "small organic molecule"; however,

Applicants 1/23/06 response (pg 12-13) includes the argument, and supporting references, that the relevant art teaches that small molecules weigh under 1000 daltons. The Examiner finds this argument persuasive and notes that the following protein kinase A inhibitors recited in claim 23 are "small organic molecules": "N-[2-((p-bromocinnamyl)amino)ethyl]-5-isoquinolinesulfonamide, 1-5(5-isoquinoline-sulfonyl)-2-methylpiperzine, KT5720, 8-bromo-cAMP and dibutyryl-CAMP. The inhibitors encompassed by Formula I in claim 24 are also "small organic molecules". However, the "PKA Heat Stable Inhibitor isoform α" recited in claim 23 is protein with a molecular weight over 1 kDa and is therefore not a small organic molecule. Similarly, the "ptc antisense construct" recited in claim 22 is not a small organic molecule (the specification teaches on pg 6 that these constructs comprise at least 20 nucleic acid residues).

In view of the species election, claims 23 and 24 will be examined in so far as they read upon the elected species of "small organic molecule". The species election requirement between small organic molecules, ptc antisense construct, and protein kinase inhibitors (that are not small organic molecules) is maintained in view of the discrete structure of these molecules and the separate status in the art.

The requirement is still deemed proper and is therefore made FINAL.

Claims 1-4 and 19-26 are under consideration, in so far as they are drawn to the species of *ptc* therapeutic that is a small organic molecule.

Withdrawn Objections and/or Rejections

The following page numbers refer to the 9/20/05 Office Action.

All objections to, and rejections of, claim 18 are moot in view of Applicants' cancellation of this claim.

The objections to the specification and drawings on pg 3-4 are *withdrawn* in view of Applicants' amendments to the specification.

The objection to claims 1-4 at pg 4 as encompassing non-elected inventions is *withdrawn* in view of Applicants' amendments to the claims.

The rejection of claims 19-21 under 35 U.S.C § 112, second paragraph, at pg 9-10 for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention is *withdrawn* in view of Applicants' persuasive arguments at pg

The rejection of claims 1-4 and 19-21 under 35 U.S.C. § 102(a) as clearly anticipated by Fujita et al (1997) is *withdrawn* in view of Applicants' amendments to the claims.

Maintained Objections and/or Rejections

Claim Objections

Claims 1, 2 and 19-22 are objected to because abbreviations (i.e., "ptc") should be spelled out in all independent claims for clarity. This objection was set forth at pg 4 of the 9/20/05 Office Action for claims 1, 2 and 19-21; new claim 22 is herewith added to this objection.

Applicants' arguments (1/23/06; pg 9) as they pertain to the objection have been fully considered but are not deemed to be persuasive for the following reasons. Applicants argue that the term "ptc therapeutic" is a defined term that is clearly described on the specification at pg 12, lines 17-22.

Applicants' arguments have been fully considered but are not found persuasive. The Examiner has fully considered the definition of "ptc therapeutic" on pg 12 of the specification. However, it is noted that definitions on pg 12 of the specification are directed to "*ptc* therapeutic" (i.e., *ptc* is italicized, unlike in the claims). If the claims were amended such that the term "*ptc* therapeutic" was used, such that it was clear that the term as used in the specification on page 12 was being referred to in the claims, the objection would be withdrawn.

Claim Rejections - 35 USC § 112, 1st paragraph

Claims 1-4 and 19-26 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. This rejection was set forth as a scope of enablement rejection for claims 1-4 and 19-21 at pg 4-7 of the 9/20/05 Office Action. However, Applicants' amendments have limited the scope of the claims such that the entire scope of the amended claims lacks enablement. New claims 22-26 are herewith included in this rejection.

Applicants' arguments (1/23/06; pg 10-11) as they pertain to the rejection have been fully considered but are not deemed to be persuasive for the following reasons.

With respect to claims 1, 3 and 4, Applicants submit that the amended claims more particularly point out the claimed subject matter. Applicants argue that the specification describes the characteristics of useful *ptc* therapeutics and how to identify such therapeutics. Applicants argue that the specification demonstrates the lack of active hedgehog in hedgehog null mice and that this result represents similar effects that can be achieved by negatively regulating hedgehog signal transduction by administering a hedgehog mutant with little or no wildtype activity, or a *ptc* therapeutic that inhibits the hedgehog/patched pathway. Applicants argue that the making and testing of a range of *ptc* therapeutics can be done without undue experimentation.

Applicants' arguments have been fully considered but are not found persuasive. Claim 1, as amended, encompasses a method for inhibiting or reducing the proliferation or growth of lung cancer cells, comprising contacting the cells with a small organic molecule that inhibits the hedgehog/patched signal transduction pathway. This method encompasses both *in vitro* inhibition of lung cancer cells (e.g., a lung cancer cell line) and *in vivo* inhibition of lung cancer cells (e.g., a lung tumor in an organism). Dependent claim 3 is limited to lung cancer cells in culture and dependent claim 4 is limited to lung cancer cells in an animal. New claims 25 and 26 depend from claim 1 and limit the method to small cell lung cancer cells or non-small cell lung cancer cells (claim 25) or adenocarcinoma, lung cell carcinoma or squamous cell carcinoma cells (claim 26).

The amount of direction or guidance provided by Applicants regarding the structure or nature of the compounds encompassed by the claimed methods is minimal. The specification provides very limited teachings regarding "small organic molecules" that are inhibitors of the hedgehog/patched signal transduction pathway: the specification teaches only that the term "*ptc* therapeutic" includes "a *hedgehog* antagonist" and can be a "small organic molecule" (pg 12); that inhibitory *ptc* therapeutics can be used to treat lung cancer, including small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC) (pg 16); that hedgehog/patched pathway antagonists, for example small organic molecules, can be identified through screening assays (pg 48); and that *Shh* gene mutation leads to improper lung formation in embryonic mice (pg 69). The specification does not provide any working examples demonstrating small organic molecules that inhibit lung cancer cell growth.

The prior art teaches that the N-terminal fragment of Sonic hedgehog (Shh-N) is capable of stimulating *in vitro* proliferation of lung squamous carcinoma cells, and that anti-Shh-N antibody inhibits proliferation (see pg 661 of Fujita et al 1997, cited in the 9/20/05 Office Action). The prior art does not teach any small organic molecule inhibitors of patched/hedgehog signaling capable of inhibiting lung cancer cell growth, either *in vitro* or *in vivo*.

In view of the limited teachings of the specification and the prior art, it would require undue experimentation to practice the claimed methods. The specification and the prior art do not identify any small organic molecules that can inhibit lung cancer cell growth or proliferation. The specification merely invites the skilled artisan to engage in further undue experimentation to screen small organic molecules *in vitro* in order to determine whether or not they have the ability to inhibit hedgehog/patched signaling. The specification provides no guidance (such as structural information) to skilled artisan regarding the identity of the small organic molecules that would work in the claimed invention. Further, even if a particular compound is identified as a hedgehog/patched pathway inhibitor, it is not predictable whether or not it will inhibit lung cancer cell growth, either *in vitro* or *in vivo*, without further screening to actually test such inhibition.

Applicants' teaching regarding the phenotype of hedgehog null mutants is not instructive as to the nature of small organic molecules that will inhibit lung cancer cell growth.

With respect to claim 2 (and dependent claims 20-24), Applicants argue that the specification provides ample support for the promotion of normal lung tissue's growth and maintenance, such that *ptc* therapeutics could be used therapeutically to promote growth and repair of damaged lung tissue. With regard to *ptc* therapeutics, Applicants argue that the specification describes hedgehog protein (and derivatives), small molecules and antisense constructs, including antagonists of patched protein which would attenuate the inhibitory effects of patched.

Applicants' arguments have been fully considered but are not found persuasive. Claim 2, as amended, encompasses a method for inducing the formation of, or the maintenance or functional performance of normal lung tissue, comprising contacting the lung tissue with a ptc therapeutic that promotes hedgehog/patched signal transduction. Formation of "lung tissue" encompasses both *in vitro* formation of lung tissue (e.g., lung cell culture or lung "explant" organ culture) and *in vivo* formation of tissue (e.g., production of adult lung tissue from either fetal lung tissue or damaged adult lung tissue). The term "normal lung tissue" excludes "abnormal lung tissue" and implies that the newly formed tissue must be functional. The term "ptc therapeutic" encompasses a variety of molecules, including the hedgehog protein as well as small organic molecules, antisense constructs and protein kinase A inhibitors as taught on pg 5-6 of the specification. Dependent claims 19, 20 and 21 limit the ptc therapeutic to a small organic molecule that binds to patched protein (claim 19) or to a therapeutic that binds to patched and mimics hedgehog (claim 20) and is small organic molecule (claim 21). New claims 22-24 depend from claim 2 and limit the ptc therapeutic to a protein kinase A inhibitor (claims 22-24) or a ptc antisense construct (claim 24).

The amount of direction or guidance provided by Applicants regarding the structure or nature of the compounds encompassed by the claimed methods is minimal.

With regard to the Sonic hedgehog (Shh) protein, it is clear from the teachings of the specification and prior art that Shh plays an important role in the development of the lung. The specification teaches that a null mutant of the *Shh* gene leads to abnormal

lung growth and formation in embryonic mice (pg 69). The prior art teaches that overexpression of Shh during development produces mice with abnormal lung tissue at birth (see pg 56 of Bellusci et al. 1997; cited on the 12/3/03 IDS). However, the relevant art also makes it clear that lung morphogenesis is extremely complex, and that Shh is only one of a large number of factors involved in this process. For example, Warburton (2000) teaches, "Lung morphogenesis is determined by functional integration of key transcriptional factors, peptide growth factor receptor-mediated signaling, extracellular matrix, integrin and non-integrin signaling. These inputs are integrated during the normal process of embryonic, fetal and postnatal lung morphogenesis. They instruct organized temporo-spatial patterns of cellular proliferation, cell lineage differentiation, cell movement and cell death that determine structure and hence physiological function (see pg 57 of Warburton et al 2000. Mechanisms of Development. 92: 55-81)." Kumar (2004) teaches, "Formation and orderly development of the mammalian lung results from a complex set of cell to cell and cell to matrix interactions following transcriptional regulation during pulmonary organogenesis. Transcriptional control of differentiation genes early on and epithelial-mesenchymal interactions mediated by growth factors later on, resulting in the formation of conducting airways and an extensive alveolar capillary interface, is critical for normal lung development" and "Epithelial-endothelial interactions during lung development are important in establishing a functional blood gas interface. Epithelial-mesenchymal interactions mediated by growth factors are also important in the restoration of normal alveolar architecture after lung injury. Further understanding of the role of these growth factors and their cellular interactions in bronchopulmonary dysplasia and in tissue repair following lung injury, may lead to development of better therapeutic modalities in treating these disorders" (see pg 464 of Kumar et al. 2004. Frontiers in Bioscience. 9: 464-480). Kumar (2004) also teaches that the *in vivo* processes of lung development and repair remained poorly understood, even as of 2004:

> "Even though it is important to study the effects of individual growth factors on isolated cell preparations, there are multiple growth factors present in vivo at different times during lung development and during the lung injury and repair process. The complex interactions among growth factors and the spatial and

> temporal relationship to cellular proliferation and differentiation might be different in vivo and is as yet unclear. The effects of a particular growth factor may be different at different sites and at different time points. The dynamic interplay among type II cells, the extracellular matrix and various growth factors may determine multicellular functions and play an important role in normal lung development and in repair of the lung epithelium following injury. A better understanding of the epithelial-endothelial interaction and regulation of the alveolar-capillary interface will provide important clues for novel therapies in preterm infants with chronic lung injury" (pg 474).

In view of these teachings, it is not predictable how contacting lung tissue with Shh, or another ptc therapeutic that stimulates hedgehog signaling, could be used to produce functional lung tissue. Contacting lung tissue with Shh alone may lead to abnormal lung tissue formation (such as observed by Bellusci, 1997, cited above), and it would require undue experimentation to determine the precise time and location at which Shh (or another ptc therapeutic) must be administered, and what other factors are required in conjunction, in order to form functional lung tissue.

Furthermore, the specification envisions other inducers of hedgehog/patched signaling that include small organic molecules, antisense constructs and protein kinase A inhibitors, but provides little guidance as to which of these molecules will actually work to induce formation of lung tissue.

The elected species of ptc therapeutic under consideration is "small organic molecule"; therefore, the enablement of claim 2 (and dependent claims 20-24) will be considered with regard to this species. The specification does not provide a definition of "small organic molecules". In view of the relevant art cited by Applicants in the 1/23/06 response, this term encompasses any organic compound of less than 1000 daltons. No structural limitations are implied by this term. Of the vast genus of molecules that would meet the limitation of "small organic molecule", the only specific types of "small organic molecules" disclosed in specification is the subgenus consisting of "protein kinase A inhibitors". However, Applicants do not provide any working examples related to the administration of protein kinase A inhibitors and "inducing the formation of, or the maintenance or functional performance of normal lung tissue". The term "normal lung tissue" is not defined in the specification and includes *in vivo* fetal or adult lung tissue,

explanted fetal or adult lung tissue (organ culture), and *in vitro* culture of isolated cells. It is not predictable whether or not protein kinase A inhibitors would stimulate formation of one or any of these types of "normal lung tissue". In fact the relevant art demonstrates the protein kinase A inhibitors inhibit differentiation of fetal lung tissue. Ballard (1991) teaches that surfactant protein is "marker for differentiated type II cells" in explant of human fetal lung (pg 2916 of Ballard, 1991. Endocrinology. 128(6): 2916-24.). Ballard further teaches that while increase cAMP correlated with an increase in surfactant protein, "exposure to H-8 and A-3, which inhibit protein kinase-A, blocked the rise in surfactant components" (pg 2922). Furthermore, indomethacin treatment blocked the increase in cAMP and "indomethacin-treated explants appeared less mature by light microscopy, resembling preculture tissue" (pg 2922). The protein kinase inhibitor H-7 used by Ballard is 1-(5-isoquinolinesulfonyl]2-methylpiperazine (see pg 1917), which is specifically recited by Applicants as a protein kinase A inhibitor in claim 23. Further, Yang (1991) teaches that in adult rat lung organ culture "cAMP acts as a positive regulator in proliferation of lung tissues" (see Abstract of Yang, 1991. Exp Lung Res. 15(4): 527-35). In view of these teachings it is not predictable how a protein kinase A inhibitor could be used to stimulate lung tissue growth. Additionally, Applicants have not provided any guidance as to the structure or nature of any other "small organic molecules" that can be used to stimulate lung tissue growth. Applicants merely invite the skilled artisan to screen a large number of small organic molecules for those that can activate hedgehog/patched signaling pathway, and then screen those molecules for a subset that can also activate the growth of lung tissue.

Finally, it is noted that claim 23 includes "8-bromo-cAMP" and "dibutyryl-cAMP" in the list of "protein kinase A inhibitors". However, it is well known in the relevant art that these analogs of cAMP are protein kinase A activators rather than inhibitors. For example, Schwede (2000) teaches that dibutyryl-cAMP is "a potent activator of both PKA-I and –II" (pg 204) and "8-Br-cAMP is known to selectively activate PKA-I and –II" (pg 205; Schwede et al. Pharmacology & Therapeutics. 2000. 199-226.) It is not predictable how a protein kinase A activator could be used as a protein kinase inhibitor.

Therefore, claim 23 lacks enablement for the species of "8-bromo-cAMP" and "dibutyryl-cAMP".

In summary, it is acknowledged that the level of skill of those in the art is high, but it is not disclosed and not predictable from the limited teachings of the prior art and specification (1) what type of small organic molecules exist that act through the hedgehog/patched pathway and can be used to inhibit lung cancer cell growth or (2) what type of ptc therapeutic exists that that act through the hedgehog/patched pathway could be used to induce the formation of functional lung tissue, and how this ptc therapeutic could be used in a method of forming functional lung tissue. The specification fails to teach the skilled artisan how to use the claimed methods without resorting to undue experimentation.

Claim Rejections - 35 USC § 112, 1st paragraph, written description

Claims 1-4 and 19-26 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This rejection was set forth at pg 7 of the 9/20/05 Office Action for claims 1-4 and 19-21; new claims 22-26 are herewith included in this rejection.

Applicants' arguments (1/23/06; pg 12) as they pertain to the rejection have been fully considered but are not deemed to be persuasive for the following reasons.

In the response, Applicants submit the specification contains examples of other than Sonic hedgehog that can be used to practice the claimed methods. Applicants point to pgs 12, 57 and 60-62. Applicants argue that compositions to be used in the claimed methods are described, and administration of the compositions is described.

Applicants' arguments have been fully considered but are not found persuasive. While Applicants have described examples of compounds that could potentially be used in the claimed method, Applicants have not described examples of compounds that

actually work in the claimed methods. Applicants' claims encompass a wide variety of "small organic molecules" without any sort of structural description of the small molecules, other than a sole example consisting of the subgenus of protein kinase A inhibitors, which for the reasons described above in the section "Claim Rejections - 35 USC § 112, 1st paragraph, enablement" are not considered an example of a compound that will work in the claimed method. For these reasons, it is maintained that Applicants have not demonstrated that they were in possession of a genus of small organic molecules that could be used in the claimed methods. As set forth previously, adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method of isolating it. The compound itself is required. See *Fiers v. Revel*, 25 USPQ2d 1601 at 1606 (CAFC 1993) and *Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016.

New Rejections necessitated by Applicants' amendments

Claim Rejections - 35 USC § 112, 2nd paragraph

Claim 23 is rejected under 35 U.S.C. § 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claim 23 is indefinite because the list of "protein kinase A inhibitors" includes "8-bromo-cAMP" and "dibutyryl-cAMP". However, it is well known in the relevant art that these analogs of cAMP are protein kinase A activators, not inhibitors. For example, Schwede (2000) teaches that dibutryryl-cAMP is "a potent activator of both PKA-I and –II" (pg 204) and "8-Br-cAMP is known to selectively activate PKA-I and –II" (pg 205; of Schwede et al, 2000, cited above).

Conclusion

No claims are allowed.

Applicants' amendments necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicants are reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Zachary C. Howard whose telephone number is 571-272-2877. The examiner can normally be reached on M-F 9:30 AM - 6:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary B. Nickol can be reached on 571-272-0835. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

zch

BRENDA BRUMBACK
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600